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Breakdown (lysis) of blood clots formed in blood vessels, using medication Medical intervention ThrombolysisAngiograph before and after thrombolytic therapy in a case of acute limb ischemia.Other namesFibrinolytic therapyMedlinePlus007089eMedicine811234[edit on Wikidata] Thrombolysis, also called fibrinolytic therapy, is the breakdown (lysis) of blood clots formed in blood vessels, using medication. It is used in ST elevation myocardial infarction, stroke, and in cases of severe venous thromboembolism (massive pulmonary embolism or extensive deep vein thrombosis).[citation needed] The main complication is bleeding (which can be dangerous), and in some situations thrombolysis may therefore be unsuitable. Thrombolysis can also play an important part in reperfusion therapy that deals specifically with blocked arteries. Diseases where thrombolysis is used: ST elevation myocardial infarction: Large trials have shown that mortality can be reduced using thrombolysis (particularly fibrinolysis) in treating heart attacks.[1] It works by stimulating secondary fibrinolysis by plasmin through infusion of analogs of tissue plasminogen activator (tPA), the protein that normally activates plasmin. Stroke: Thrombolysis reduces major disability or death when given within 3 hours (or perhaps even 6 hours) of ischaemic stroke onset when there are no contraindications to treatment.[2][3][4] Massive pulmonary embolism. For the treatment of a massive pulmonary embolism, catheter-directed therapy is a safer and more effective alternative to systemic thrombolysis. This involves the injecting of drugs directly into the clot.[5] Severe deep vein thrombosis (DVT), such as phlegmasia cerulea dolens, which threatens limb loss, or iliofemoral DVT, where clots involve at a minimum the common iliac vein[6] Acute limb ischaemia[7] Clotted hemothorax[8] Thrombolysis is usually intravenous. It may also be used directly into the affected blood vessel during an angiogram (intra-arterial thrombolysis), e.g. when patients present with stroke beyond three hours or in severe deep vein thrombosis (catheter-directed thrombolysis).[9] Thrombolysis is performed by many types of medical specialists, including interventional radiologists, vascular surgeons, cardiologists, interventional neuroradiologists, and neurosurgeons. In some countries such as the United States of America, emergency medical technicians may administer thrombolytics for heart attacks in prehospital settings, by on-line medical direction. In countries with more extensive and independent qualifications, prehospital thrombolysis (fibrinolysis) may be initiated by the emergency care practitioner (ECP). Other countries which employ ECP's include, South Africa, the United Kingdom, and New Zealand. Prehospital thrombolysis is always the result of a risk-benefit calculation of the heart attack, thrombolysis risks, and primary percutaneous coronary intervention (pPCI) availability.[citation needed] Thrombolysis is not without risks. Therefore, clinicians must select patients who are to be best suited for the procedure, and those who have the least risk of having a fatal complication. An absolute contraindication is in itself enough to avoid thrombolysis, while a relative contraindication needs to be considered in relation to the overall clinical situation.[citation needed] Absolute contraindications:[10] Any previous history of hemorrhagic stroke, ischemic stroke within 3 months. History of stroke, dementia, or central nervous system damage within 1 year Head trauma within 3 weeks or brain surgery within 6 months Known intracranial neoplasm Suspected aortic dissection Internal bleeding within 6 weeks Active bleeding or known bleeding disorder Traumatic cardiopulmonary resuscitation within 3 weeks Relative contraindications:[10] Oral anticoagulant therapy Acute pancreatitis Pregnancy or within 1 week postpartum Active peptic ulceration Transient ischemic attack within 6 months Dementia Infective endocarditis Active cavitating pulmonary tuberculosis Advanced liver disease Intracardiac thrombi Uncontrolled hypertension (systolic blood pressure >180 mm Hg, diastolic blood pressure >110 mm Hg) Puncture of noncompressible blood vessel within 2 weeks Previous streptokinase therapy Major surgery, trauma, or bleeding within 2 weeks Absolute contraindications:[11][12] Uncertainty about time of stroke onset (e.g. patients awakening from sleep). Coma or severe obtundation with fixed eye deviation and complete hemiplegia. Hypertension: systolic blood pressure ≥ 185mmHg; or diastolic blood pressure >110mmHg on repeated measures prior to study (if reversed, patient can be treated). Clinical presentation suggestive of subarachnoid haemorrhage even if the CT scan is normal. Presumed septic embolus. Patient having received a heparin medication within the last 48 hours and has an elevated Activated Prothrombin Time (APTT) or has a known hereditary or acquired haemorrhagic diathesis INR >1.7 Known advanced liver disease, advanced right heart failure, or anticoagulation, and INR > 1.5 (no need to wait for INR result in the absence of the former three conditions). Known platelet count 22.0 mmol/L. Relative contraindications:[13] Severe neurological impairment with NIHSS score >22. Age >80 years. CT evidence of extensive middle cerebral artery (MCA) territory infarction (sulcal effacement or blurring of grey-white junction in greater than 1/3 of MCA territory). Stroke or serious head trauma within the past three months where the risks of bleeding are considered to outweigh the benefits of therapy. Major surgery within the last 14 days (consider intra-arterial thrombolysis). Patient has a known history of intracranial haemorrhage, subarachnoid haemorrhage, known intracranial arteriovenous malformation or previously known intracranial neoplasm Suspected recent (within 30 days) myocardial infarction. Recent (within 30 days) biopsy of a parenchymal organ or surgery that, in the opinion of the responsible clinician, would increase the risk of unmanageable (e.g. uncontrolled by local pressure) bleeding. Recent (within 30 days) trauma with internal injuries or ulcerative wounds. Gastrointestinal or urinary tract haemorrhage within the last 30 days or any active or recent haemorrhage that, in the opinion of the responsible clinician, would increase the risk of unmanageable (e.g. by local pressure) bleeding. Arterial puncture at non-compressible site within the last 7 days. Concomitant serious, advanced or terminal illness or any other condition that, in the opinion of the responsible clinician would pose an unacceptable risk. Minor or Rapidly improving deficit. Seizure: If the presenting neurological deficit is deemed due to a seizure. Pregnancy is not an absolute contraindication. Consider intra-arterial thrombolysis. Hemorrhagic stroke is a rare but serious complication of thrombolytic therapy. If a patient has had thrombolysis before, an allergy against the thrombolytic drug may have developed (especially after streptokinase). If the symptoms are mild, the infusion is stopped and the patient is commenced on an antihistamine before infusion is recommenced. Anaphylaxis generally requires immediate cessation of thrombolysis.[citation needed] Thrombolysis therapy uses thrombolytic drugs that dissolve blood clots. Most of these drugs target fibrin (one of the main constituent of blood clots) and are therefore called fibrinolytics. All currently approved thrombolytic drugs are biologics, either derived from Streptococcus species, or, more recently, using recombinant biotechnology whereby tPA is manufactured using cell culture, resulting in a recombinant tissue plasminogen activator or rtPA.[citation needed] Some fibrinolytics are: Streptokinase (Kabikinase)[14] Urokinase[15] Recombinant tissue plasminogen activators (rtPA) Alteplase (Activase or Actilyse)[14] Reteplase (Retavase)[16] Tenecteplase[16] Anistreplase (Eminase)[14] A 2023 meta-analysis of 44 studies[17] compared treatments for pulmonary embolism including thrombolytic therapy delivered through a catheter. Catheter-directed thrombolysis (CDT) methods included fragmentation and ultrasound use. CDT was associated with better outcomes than anticoagulation alone or systemic thrombolysis, but the studies were mostly small and observational. In people who receive CDT, there is a risk of hemorrhage as a side effect. Scientists have studied whether measuring fibrinogen in blood can be used as a biomarker to predict hemorrhage. As of 2017 it was not known if this works or not.[18] Researchers showed a 10-fold variation in the proportion of patients who received thrombolysis after stroke in England and Wales, ranging from 1 in 50 (2%) to 1 in 4 (24%). The team also showed that most of the variation was explained by hospital processes (such as how quickly people can have a brain scan) and in doctors' decision-making (who they think should or should not receive thrombolysis) rather than knowledge of the time of stroke.[19][20] Prospective, randomized clinical trials to evaluate the utility of catheter-directed thrombolysis in pulmonary embolism include HI-PEITHO (Higher-Risk Pulmonary Embolism Thrombolysis).[21] TIMI – thrombolysis in myocardial infarction ^ "Indications for fibrinolytic therapy in suspected acute myocardial infarction: collaborative overview of early mortality and major morbidity results from all randomised trials of more than 1000 patients. Fibrinolytic Therapy Trialists' (FTT) Collaborative Group". Lancet. 343 (8893): 311–22. 5 February 1994. doi:10.1016/s0140-6736(94)91161-4. PMID 7905143. ^ Wardlaw JM, Murray V, Berge E, Del Zoppo GJ (2014). "Thrombolysis for acute ischaemic stroke". 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"Ultrasound-facilitated, catheter-directed thrombolysis vs anticoagulation alone for acute intermediate-high-risk pulmonary embolism: Rationale and design of the HI-PEITHO study". Am Heart J. 251: 43–53. doi:10.1016/j.ahj.2022.05.011. hdl:1887/3494555. PMID 35588898. Retrieved from " Welcome! You're still at Nurseslabs.com—just with a new look! Our mission hasn't changed: from exam prep and reliable care plans to expert tips and uplifting success stories, we remain your steadfast ally in all things nursing. Count on us for precise, easy-to-digest resources so you can keep delivering exceptional care and making a real difference. Introducing our comprehensive guide to crafting your own nursing care plan. It comes with a complimentary collection of nursing diagnosis examples and care plans, perfect for both student nurses and seasoned professionals. Empower your Medical-Surgical Nursing practice with our expertly curated study guides. Master key concepts, patient care strategies, and perioperative techniques to boost your skills and exam readiness. View All Editor's Picks Form of hormonal contraception combining both an estrogen and a progestogen Combined hormonal contraception Combined oral contraceptive pill Combined contraceptive patch Combined contraceptive vaginal ring BackgroundTypeHormonalFirst use oral pill - 1960 injection – 1960s patch – 2003 vaginal ring – 2009 Failure rates (first year)Perfect use0.3[1]%Typical use9[1]%UsageReversibilityon discontinuationUser reminders?Advantages and disadvantagesSTI protectionNoPeriodsTypically regular and lighterWeightNo evidence of weight gain[1] Combined hormonal contraception (CHC), or combined birth control, is a form of hormonal contraception which combines both an estrogen and a progestogen in varying formulations.[1][2] The different types available include the pill, the patch and the vaginal ring, which are all widely available,[3] and an injection, which is available in only some countries.[4] They work by mainly suppressing luteinising hormone (LH) and follicle-stimulating hormone (FSH) and in turn preventing ovulation.[1] The pill, patch, and vaginal ring are all about 93% effective with typical use.[5] Beneficial health effects include reduced risks of ovarian, endometrial and colorectal cancers. CHC can also provide improved control of some menstrual problems. Adverse effects include a small but higher risk of venous thromboembolism, arterial thromboembolism, breast cancer and cervical cancer.[4][6] With perfect use, less than 1% of women will become pregnant during the first year of using CHC. However, with typical use 9% of women will become pregnant during the first year.[7] Traditionally, to mimic a normal menstrual cycle, CHC is used for 21 consecutive days. For all of these methods (pill, patch, vaginal ring), these 21 days are typically followed by either 7 days of no use (for the pill, patch or vaginal ring) or 7 days of administration of placebo pills (for the pill only). During these 7 days, withdrawal bleeding occurs. For those women who do not desire withdrawal bleeding or require bleeding to be suppressed completely, medication regimens can be tailored to the individual with extended periods of use and infrequent hormone-free periods. The efficacy of CHC is the same whether these methods are used continuously or with a 7-day break to allow for withdrawal bleeding.[8] Combined oral contraceptives (COCs) can be used to treat menstrual cycle disorders including heavy menstrual bleeding,[9] and pelvic pain disorders such as endometriosis[10] and dysmenorrhea.[11] CHCs are also a first line treatment for polycystic ovary syndrome for menstrual abnormalities, acne, and hirsutism.[12] Perimenopausal women on combined oral contraceptives have increased bone density,[13] and COCs can be used to decrease hot flashes.[14] Combined oral contraceptives have been shown to reduce risk of endometrial cancer, BRCA1 and BRCA2 ovarian cancer, and a modest reduction in colon cancer.[14][15] Types of Combined Hormonal Contraceptives Formulation Efficacy Perfect Use Combined oral contraceptive pill[7] Various formulations (10-50 µg estrogen (average 20–35)[16] and 0.05–3 mg progesterone[17]) 9% failure rate with typical use (method not used consistently or correctly) 0.3% failure rate with perfect use [7][18] Meant to be taken at the same time every day (some pills can be taken within 2–24 hours and still be effective)[19] Combined contraceptive patch[7] 120-150 µg of norelgestromin and 20-35 µg ethinyl estradiol daily[20][21][22] New patch used once a week, after 3 weeks patch is not worn to allow for withdrawal bleeding[19] Combined contraceptive vaginal ring[7] 120-150 µg etonogestrel and 13-15 µg ethinyl estradiol daily[20][23][24] Vaginal ring worn for 21 days and removed for the following 7 days to allow for withdrawal bleeding[19] Combined injectable contraceptive,[25] additional category of CHC not available in the USA or UK[26] The Faculty of Sexual and Reproductive Healthcare has issued guidelines for incorrect use[1] Hormonal use for 21 days followed by 7 day withdrawal is the most common regimen, however schedules are variable. Other factors affecting effectiveness include drug interactions, malabsorption and body weight.[27] Hypothalamic-pituitary-adrenal axis Prevention of ovulation occurs via inhibition of the hypothalamic–pituitary–gonadal axis, through progesterone and estrogen providing negative feedback to the hypothalamus and inhibiting the production of gonadotropin releasing hormone (GnRH). GnRH typically promotes the release of LH and FSH from the pituitary. The presence of estrogen in CHCs results in downstream inhibition of luteinizing hormone (LH) and follicular stimulating hormone (FSH) which typically act at the ovarian level to induce ovulation and promote development of the follicle respectively.[28] Progesterone also contributes to the contraceptive effect by making changes to the cervical mucus, endometrium and tubal motility.[28] Although the risk of venous thromboembolism, arterial thromboembolism, breast cancer and cervical cancer in CHC users is small, all CHCs are associated with higher risks of these compared to no use. Given that the vast majority of the studies evaluating these associations have been observational studies, causation between CHC use and these conditions is unable to be determined.[29][30] All CHCs are associated with an increased incidence of venous and arterial thromboembolism. However, those containing higher doses of estrogen are associated with an increase in venous and arterial thromboembolism.[31][32] In addition, some formulations of progesterone, including gestodene, desogestrel, cyproterone acetate and drospirenone, in combination with estrogen, have been associated with higher rates of venous thromboembolism compared to formulations containing a type of progesterone called levonorgestrel.[33] Other adverse effects include nausea, headaches, breast pain, skin pigmentation, irregular menstrual bleeding, absent periods and irritation from contact lenses. Changes in libido and mood, decline of liver function and raised blood pressure may also occur.[1] vte Risk of venous thromboembolism (VTE) with hormone therapy and birth control (QResearch/CPRD) Type Route Medications Odds ratio (95% CITooltip confidence interval) Menopausal hormone therapy Oral Estradiol alone ≤1 mg/day >1 mg/day 1.27 (1.16–1.39)*1.22 (1.09–1.37)*1.35 (1.18–1.55)* Conjugated estrogens alone ≤0.625 mg/day >0.625 mg/day 1.49 (1.39–1.60)*1.40 (1.28–1.53)*1.71 (1.51–1.93)* Estradiol/medroxyprogesterone acetate 1.44 (1.09–1.89)* Estradiol/dydrogesterone ≤1 mg/day E2 >1 mg/day E2 1.18 (0.98–1.42)1.12 (0.90–1.40)1.34 (0.94–1.90) Estradiol/norethisterone ≤1 mg/day E2 >1 mg/day E2 1.68 (1.57–1.80)*1.38 (1.23–1.56)*1.84 (1.69–2.00)* Estradiol/norgestrel or estradiol/drospirenone 1.42 (1.00–2.03) Conjugated estrogens/medroxyprogesterone acetate 2.10 (1.92–2.31)* Conjugated estrogens/norgestrel ≤0.625 mg/day CEEs >0.625 mg/day CEEs 1.73 (1.57–1.91)*1.53 (1.36–1.72)*2.38 (1.99–2.85)* Tibolone alone 1.02 (0.90–1.15) Raloxifene alone 1.49 (1.24–1.79)* Transdermal Estradiol alone ≤50 µg/day >50 µg/day 0.96 (0.88–1.04)0.94 (0.85–1.03)1.05 (0.88–1.24) Estradiol/progestogen 0.88 (0.73–1.01) Vaginal Estradiol alone 0.84 (0.73–0.97) Conjugated estrogens alone 1.04 (0.76–1.43) Combined birth control Oral Ethinylestradiol/norethisterone 2.56 (2.15–3.06)* Ethinylestradiol/levonorgestrel 2.38 (2.18–2.59)* Ethinylestradiol/norgestimate 2.53 (2.17–2.96)* Ethinylestradiol/desogestrel 4.28 (3.66–5.01)* Ethinylestradiol/gestodene 3.64 (3.00–4.43)* Ethinylestradiol/drospirenone 4.12 (3.43–4.96)* Ethinylestradiol/cyproterone acetate 4.27 (3.57–5.11)* Notes: (1) Nested case–control studies (2015, 2019) based on data from the QResearch and Clinical Practice Research Datalink (CPRD) databases. (2) Bioidentical progesterone was not included, but is known to be associated with no additional risk relative to estrogen alone. Footnotes: * = Statistically significant (p < 0.01). Sources: See template. The estrogen in combined hormonal contraception can increase the risk of blood clotting in some women. In particular, this can manifest as a deep vein thrombosis or pulmonary embolism. However, the risk with low-dose combined hormonal contraceptives remain relatively low in most cases. Health providers may recommend against formulations with estrogen in women with certain risk factors including personal or family history of blood clots, pregnancy and the first 3 weeks postpartum, obesity, inactivity, and coagulation disorders.[34][35] Additionally, combined hormonal contraceptives are sometimes not recommended in the first 4–6 weeks postpartum after delivery due to concerns of effect on breastfeeding performance.[35] Estrogens and progestins are metabolized in the liver, so there is a theoretical concern for use in women with liver disease.[35] Large studies have shown a slight increased incidence of breast cancer among hormonal contraceptive users compared to nonusers.[36] However, the overall risk of breast cancer in users and nonusers remains low.[36] Research has also shown a link between cervical cancer and long-term use of combined hormonal contraception, particularly in women with chronic HPV infection of the cervix.[37] Combined hormonal contraceptives are also associated with a decreased risk of endometrial, ovarian, and colorectal cancers.[38] The most common side-effects of combined hormonal contraceptives include headache, nausea, breast tenderness, and breakthrough bleeding. Vaginal ring use can include additional side-effects including vaginal irritation and vaginal discharge. Contraceptive skin patch use can also include a side-effect of skin irritation around the patch site.[39] Breakthrough bleeding within the first 3–6 months is generally not harmful and often resolves with persistent use.[35] Contradictory research exists on the effects of combined hormonal contraceptives on weight gain. Clinical studies have shown some women report weight gain while others report weight loss. Several mechanisms for weight gain have been theorized including increased fluid retention, increase in muscle tissue, and increase in body fat. Many women stop taking combined hormonal contraceptives because they are concerned about weight gain; however, the link remains uncertain.[40] The effect of combined hormonal contraceptives on mood is unclear at this point. There have been some large cohort studies suggesting there may be an association with mood-related side-effects. Patient-perceived changes in mood remain one of the most common reasons for hormonal contraceptive discontinuation.[41] Further information: Progestogen (medication) § Mood changes Medications that induce liver enzymes increase the metabolism of oestradiol and progestogens and subsequently may reduce the effectiveness of CHC. The advice on CHC also depends on whether the liver inducing drug is used short term, for less than two months, or long term, for more than two months.[1] Should a woman have taken ulipristal acetate (ellaOne) for emergency contraception, restarting CHC may reduce ellaOne's effectiveness, hence advice is to wait five days before commencing CHC.[1] Extra contraceptive precautions are not necessary when using CHC in combination with antibiotics that do not induce liver enzymes, unless the antibiotics cause vomiting and/or diarrhoea.[1] Medications used in the treatment of epilepsy can interact with the combined pill, patch or vaginal ring,[42] resulting in both pregnancy and shift in seizure threshold.[43] Based on a study of 16 women using oral CHC 30 µg ethinyloestradiol/150 µg levonorgestrel and anti-epileptic drug lamotrigine for 6 weeks, it was revealed that the contraceptive effectiveness could be lowered, despite lamotrigine not being an enzyme inducer.[1] An assessment of risks versus benefits of CHC is recommended in women on lamotrigine who are considering CHC, as the seizure threshold in someone on lamotrigine may be lowered by the oestrogen in CHC. In a similar mechanism, stopping CHC in a patient on lamotrigine can cause lamotrigine toxicity.[1] Long-acting reversible contraception instead may be preferable.[42] Following childbirth, the use of CHC depends on factors such as whether the mother is breastfeeding and whether she has other medical conditions including superficial venous thrombosis and dyslipidaemia.[44] When considering CHC use by age only, use is unrestricted between menarche and age 40, and can be generally used after age 40.[2] If someone is less than 21 days postpartum, they should not use combined hormonal contraceptives, according to the Centers for Disease Control and Prevention. If someone is postpartum and they also have risk factors for venous thrombosis, they should wait 21-42 days before using CHC. Within 42 days postpartum, all patients can use CHC.[45] CHC should not be used by breastfeeding women in the first six weeks after delivery and are generally not recommended in the first six months after delivery if still breastfeeding. After six months, breastfeeding women may use CHC.[46] Between 2015 and 2017, 64.9% of women ages 15–49 in the United States were using contraception, and of those 12.6% were using the oral contraceptive pill.[47] There are approximately 100 million users of combined oral contraceptives worldwide, with use being more common in Western Europe, Northern Europe, and the United States.[48] In the UK, one survey demonstrated that in 2010–2012, more than 33% of women aged 16–44 years had used oral contraception in the previous year and that it was mostly the combined type.[1] Between 2006 and 2010 only 10% of women in the US had used the contraceptive patch, and 6% had used the vaginal ring. Combined injectables are most common in China, South-East Asia and South America.[48] CHC has been used worldwide for more than 60 years,[1] with the first clinical trials on oral CHC beginning in 1956.[49] The FDA first approved the oral contraceptive in 1960. The first oral contraceptive contained 100 to 175 µg of estrogen and 10 mg of progesterone. However, at these levels significant adverse effects were seen and modern preparations contain lower levels of 30 to 50 µg of estrogen and 0.3 to 1 mg of progesterone.[50] The first contraception vaginal ring was approved for use in the United States in 2001. Development of the ring has led to several designs with different sizes, ring materials, and steroid formulations. Modern designs are made of plastic polymer rings containing sex steroids which diffuse out of the ring directly into the vaginal epithelium and into systemic circulation.[51] The first birth control patch, "Ortho Evra" was first introduced in 2002.[52] In 2014, a generic version of Ortho Evra was released and called "Xulane".[53] In 2020, the FDA approved Twirla, a low-dose transdermal combined hormonal contraceptive.[54] Progestogen-only contraception Combined oral contraceptive pill Contraceptive patch Contraceptive vaginal ring ^ a b c d e f g h i j k l m n "FSRH Clinical Guideline: Combined Hormonal Contraception (January 2019, Amended February 2019) – Faculty of Sexual and Reproductive Healthcare". www.fsrh.org. Retrieved 22 July 2019. ^ a b Altshuler, Anna L.; Gaffield, Mary E.; Kiarie, James N. (December 2015). "The WHO's medical eligibility criteria for contraceptive use: 20 years of global guidance". 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